

Nasal rhinosporidiosis in a mule

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Abstract – A mass was removed from the nostril of a mule that exhibited unilateral epistaxis and nasal discharge. Impression smears revealed oval structures consistent with spores of *Rhinosporidium seeberi*. Microscopically, the mass was composed of fibrovascular granulosomatous tissue containing sporangia *R. seeberi*. Surgical excision and antifungal treatment proved curative.

Résumé – **Rhinosporidiose nasale chez une mule.** Une masse a été extirpée de la narine d'une mule montrant de l'épistaxis unilatérale et un écoulement nasal. La technique de prélèvement par empreinte directe a révélé des structures ovales compatibles avec des spores de *Rhinosporidium seeberi*. À la microscopie, la masse était composée de tissu granulosomateux fibrovasculaire contenant des sporanges de *R. seeberi*. Une excision chirurgicale et un traitement antifongique ont guéri la maladie.

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Rhinosporidiosis is a chronic infection caused by *Rhinosporidium seeberi* that is typically found in the upper respiratory tract mucosa of domestic animals and humans with granulomatous rhinitis (1–2). Human infections occur sporadically worldwide but are most commonly reported from tropical endemic areas, such as India, Sri Lanka, and Argentina (2). In veterinary medicine, rhinosporidiosis has been reported worldwide in horses and dogs, and to a much lesser extent, in cattle, cats, foxes, and avian species (3). Although rhinosporidiosis has been well documented in horses (4), to our knowledge, it has never been described in mules. The objective of the report is to describe the clinical and pathological findings of rhinosporidiosis in a mule.

A mature (age unknown) female mule originating from the dried Pacific forest of Costa Rica had been moved 6 y previously to the humid wet forest in the Cartago region. At the time of presentation, the mule was a working animal and the owner had noted a mild but recurrent nose bleed, particularly after strenuous exercise. Subsequent to these episodes of epistaxis, the mule appeared fatigued and out of breath during routine work. The mule was examined by a local veterinarian, when a large exophytic mass arising from the mucosa was noted in the right half of the nasal cavity (Figure 1). There was also a mucopurulent discharge from the affected nostril. No other clinical abnormalities were noted.

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Figure 1. Adult mule showing a multilobulated exophytic mass arising from the nasal mucosa.

The pedunculated mass was removed by an excisional biopsy and impression smears were made and stained with May Grunwald Giemsa stain. The remaining tissue was fixed in 10% buffered formalin and submitted for histopathological examination. Cytological examination of the impression smear revealed the presence of abundant squamous cells, numerous neutrophils admixed with coccobacillary organisms, and a few well-delineated oval structures with thick walls that were consistent with spores of *Rhinosporidium seeberi*.

Microscopically, the polypoid mass was formed by a thick core of mature fibrovascular tissue lined superficially by a notably hyperplastic epithelium (Figure 2). The fibrous stroma was diffusely infiltrated with neutrophils, lymphocytes, plasma cells, and macrophages. Deeply embedded in this granulation tissue and in the submucosa, there were conspicuous spherules (sporangia) of variable size (100–300 µm in diameter), surrounded by macrophages, lymphocytes, and plasma cells (Figure 2). The spherules had a thin and weakly periodic acid-Schiff (PAS)

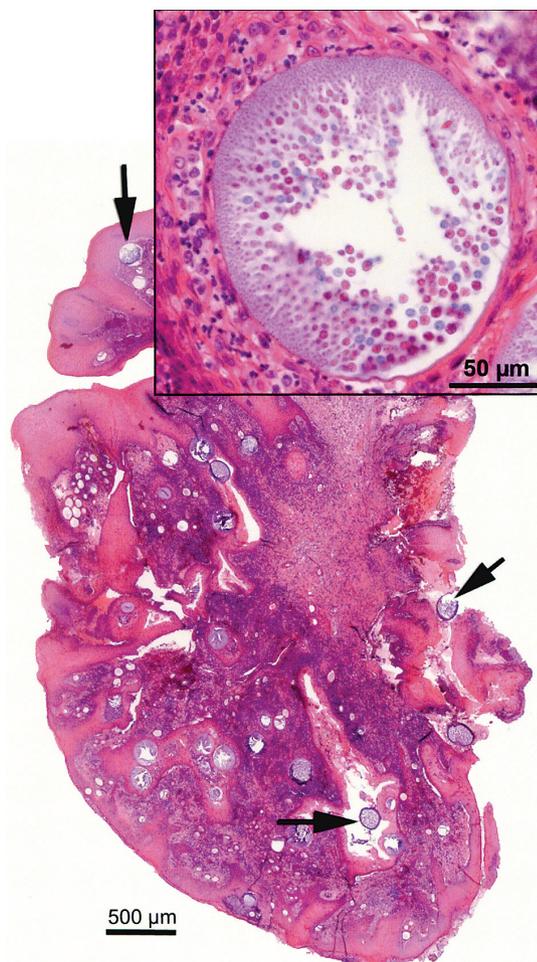


Figure 2. Note exophytic mass composed of a core of fibrovascular tissue that is superficially lined by hyperplastic mucosa. The submucosa and stromal tissues contain several large sporangia (arrows). Insert: Sporangium filled with a myriad of endospores of *Rhinosporidium seeberi*. Hematoxylin and eosin.

stain-positive wall containing a myriad of smaller (5–10 µm in diameter), strongly PAS-positive oval spores. The sporangia and the PAS-positive spores were morphologically consistent with those of *R. seeberi* (3,4). Based on the gross, cytological, and histological findings, a final diagnosis of rhinosporidiosis was made.

Since culture of *R. seeberi* is not yet possible, diagnosis of rhinosporidiosis is primarily made by observing the distinctive morphological features of *R. seeberi* in affected tissues (3). The typical lesion in humans and animals consists of a large exophytic polypoid mass arising from the nasal mucosa. Extranasal sites, such as conjunctiva, oropharynx, vagina, and bones, have been reported sporadically in human rhinosporidiosis (2).

The pathogenesis of rhinosporidiosis is poorly understood and, for many years, it has been controversial. It has been postulated that infection starts when a lacerated mucosa comes into contact with stagnant water or soil contaminated with *R. seeberi* (1). The phylogenetic classification of *R. seeberi* has also been controversial for many years, but results from recent studies with extraction DNA analysis suggest that this pathogen is a protostistan (protistan) aquatic organism belonging to the

class Mesomycetozoa and not a fungus or protozoa, as previously thought (5–6). This novel class Mesomycetozoa comprises 2 phylogenetic orders: The order Dermocystida, which includes pathogens of fish, mammals, and birds; and the order Ichthyophonida, which can be saprophytic (saprobic) or pathogenic to fish, amphibians, and insects (6,7). *Rhinosporidium seeberi* belongs to the order Dermocystida, but it still remains uncertain if the same strain infects both humans and animals. A recent study suggests that there may be multiple host-specific strains for the genus *Rhinosporidium* (8).

Although rhinosporidiosis is not a fatal disease, it may cause severe respiratory problems due to airflow obstruction, as was the case in this mule. It has clinical and pathological manifestations similar to those observed in equine infections caused by fungi such as *Coccidioides immitis*, *Cryptococcus neoformans*, and *Conidiobolus coronatus* (4,9). This latter fungus is responsible for rhinophycomycosis, the most common cause of fungal rhinitis worldwide, and it has been reported in horses with granulomatous rhinitis in Costa Rica (10). Cytologic and histopathologic examinations are always necessary to differentiate rhinosporidiosis from mycotic infections, equine nasal amyloidosis, and nasal tumors (3,4).

To our knowledge, this is the first case of rhinosporidiosis reported in a mule. The clinical and pathologic findings were identical to those typically reported for horses. As reported for rhinosporidiosis in humans and animals (1,2), surgical excision was followed by administration of fluconazole (Diflucan; Pfizer Roerig, New York, New York, USA), 0.125 mg/kg BW, IM, q1wk for 4 wk. The surgical removal combined with antifungal injections proved curative and the mule was reported by the owner to be working and in good health 22 mo after surgery.

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